Amendments to the Claims

This listing of claims will replace all prior versions and listing of claims in the application:

CLAIMS

1. (currently amended) Non-human transgenic animal model ~~of Alzheimer's disease~~ which exhibits ~~both~~ amyloid plaques and mitochondrial dysfunction.

2. (original) Animal model according to Claim 1, characterized in that it coexpresses the β-amyloid peptide precursor (APP) and a presenilin, preferably presenilin 1 (PS1).

3. (original) Animal model according to Claim 2, characterized in that it coexpresses mutated forms of APP and/or PS1.

4. (currently amended) Animal model according to Claim 3, characterized in that the mutation in the APP gene is selected from the ~~"Swedisch"~~ "Swedish", "London" and "Dutch" mutations, taken on their own or in combination.

5. (original) Animal model according to Claim 3, characterized in that the mutation in the PS1 gene is selected from the M146L, A246E, C410Y, H163R, L286V and L235P mutations, taken on their own or in combination.

6. (original) Animal model according to Claim 5, characterized in that it is the M146L mutation.

7. (original) Animal model according to Claim 1, characterized in that the mitochondrial dysfunction is an alteration, a modification, an overexpression or an inhibition of the expression of the mitochondrial proteins.

8. (original) Animal model according to Claim 7, characterized in that the proteins are intramitochondrial proteins.

9. (original) Model according to Claim 8, characterized in that the proteins are the Bax and/or cytochrome c proteins.

10. (previously presented) A method for identifying compounds which can be used for treating neurodegenerative diseases comprising exposing said compounds to the animal model of any one of claims 1 to 9.

11 - 12. (canceled)

13. (previously presented) The method of claim 10, wherein said neurodegenerative disease is Alzheimer's disease.

14. (canceled)

15. (new) Non-human transgenic animal model which exhibits amyloid plaques and neuronal death.

16. (new) Non-human transgenic animal model that exhibits amyloid plaques, neuronal loss and mitochondrial dysfunction, said model comprising:

a nucleic acid sequence encoding a mutation in the murine presenilin 1 protein comprising a M146L mutation; and

a nucleic acid sequence encoding mutations in the human β-amyloid peptide protein precursor comprising the Swedish, Dutch and London mutations.